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Case Report
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Cerebral Hyperperfusion Syndrome after Surgical Elimination of an Iatrogenic Carotid Jugular Fistula Lasting 3 Weeks Case Report

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Abstract

Background: Carotid jugular fistula is not a common complication of Hemodialysis access that often requires surgical repair. Cerebral hyperperfusion syndrome could happen after long standing carotid lesion.

Case Report: We report a case of an arteriovenous fistula that presented as dizziness and headache following a trial of jugular vein catheterization. The surgical therapy was accomplished but the patient developed major neurological deficit effects on the third day postoperative, with normal laboratory and imaging investigation; CT brain and color coated duplex ultrasonography on the carotid system.

Conclusion: Carotid jugular fistula is a rare complication of IJV catheterization. Cerebral hyperperfusion syndrome could happen after CJF elimination whatever its duration that can be reversible with early recognition and proper management.

Keywords: Carotid jugular fistula; Catheter; Complications; Fistula

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Introduction

Arteriovenous Fistulas (AVFs) can be either congenital or acquired, congenital AVFs are less common and frequently have numerous small arteriovenous connections, meanwhile acquired ones consist of a single larger connection, and they are most frequently the result of penetrating trauma or iatrogenic action (IJV catheter placements) [1-2]. Acquired AVFs have often traumatic origin, and war and after-war periods have offered, involving all the body districts arteries with prevalence in lower extremities (49%) and head and neck (29%) [3]. Ultimately, they could result with IJV catheterization for central venous pressure monitoring, parenteral nutrition or vascular access for hemodialysis [4]. Cerebral Hyperperfusion Syndrome (CHS) is a series of neurological deficits caused by postoperative high blood flow cerebral perfusion after the revascularization surgery for Carotid surgery. It happens at 50% of Moyamoya diseased patients after bypass surgery. CHS clinical symptoms are headache, aphasia, paresis, hemiparesis palsy, dysarthria and intra-cerebral hemorrhage. Most of these symptoms may completely resolve with no permanent brain injury [5].

Case Presentation

In September 2019, a 59-years-old male with ESRD on regular hemodialysis with a brachiocephalic arteriovenous on left upper limb since 5 weeks after his renal graft got failed. He was referred to our department with dizziness and headache, on examination of the neck there was no visible or palpable swelling, and the left side of the neck was warm with a continuous thrill. There was no cardiac failure or other cardiopathy, no peripheral edema, and the serum biochemical levels were all found to be within normal levels color coated Doppler showed a dilated left internal jugular vein with arterial flow inside and high peak of systolic velocity with turbulence of color without any stenosis of carotid arteries. Magnetic resonance imaging revealed venous congestion, a dilated right internal jugular vein, and the presence of a fistula between the common carotid artery and the internal jugular vein, there was no sign of cerebral ischemia (Figure 1).

The patient had undergone central vein catheterization at the neck for urgent hemodialysis after confirmation of renal graft failure 9 weeks ago. We offered both surgical and endovascular therapy to the patient. Despite the minimally invasive endovascular approaches the patient picked the surgical

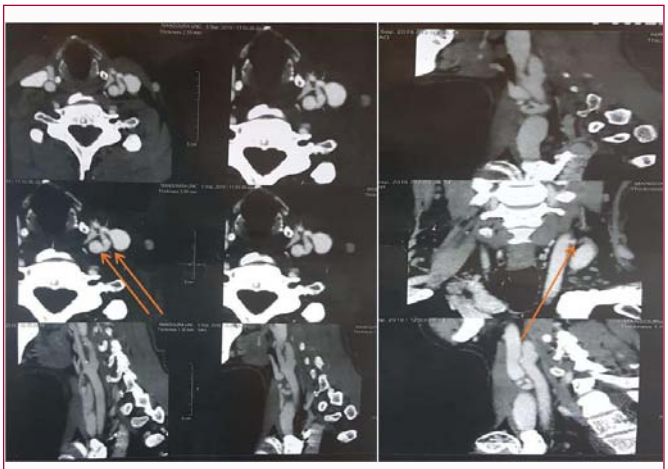

Figure 1: CT angiography of the carotid system: Evidence of arterio-venous fistula between left common carotid artery and left internal jugular vein (Arrow) 2 cm proximal to carotid bulb. The fistulous tract is tortuous (s shaped) (Double arrows). Its caliber is about 4 mm and it is (18 mm) length. No evidence of associated hematoma, leak, thrombus, aneurysmal dilatation or dissection.

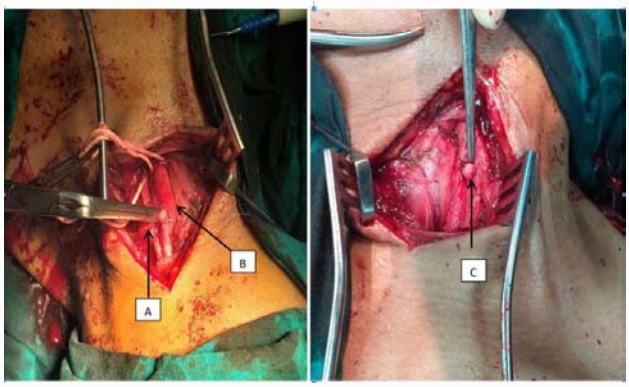

Figure 2: The fistula tract was first clamped then the fistula opening (C) was closed using hemostatic single points with 6/0 polypropylene monofilament on the two faced sides of internal jugular vein, looped by elastic rubber) (B) and common carotid artery (A).

one. The patient underwent surgical repair of the arterio-venous communication with standard approach under general anesthesia, but vessel clamping and arterial reconstruction was not needed. The fistula opening was closed using hemostatic single points with 6/0 polypropylene monofilament on the two faced sides of internal jugular vein and common carotid artery (Figure 2).

Postoperatively the patient reported complete resolution of headaches and dizziness. However, on the third day, the patient developed disturbed conscious level with chorea. The lab investigations were within the normal scale. The CT brain revealed neither infarction nor hemorrhage that did not explain the neurological deficit. Duplex scanning performed showed absence of turbulence of color Doppler and normal carotid system. The patient improved within 4 weeks and completely and all neurological findings disappeared months ago.

Discussion

Spontaneous carotid jugular fistulae are uncommon. A high index of suspicion is necessary for the diagnosis especially in absence of pulsatile mass presentation. Unlike arteriovenous fistula in the limbs; carotid jugular fistula are particularly prone to complications such as intractable high output cardiac failure, atrial fibrillation and embolization. The development of high cardiac output and heart failure depends on the proximity of the fistula to the heart and the degree of left to right shunting; determined by the diameter of the fistula and the number of fistulae present. So in our case the presentation of high cardiac output failure not reported as the fistula was single, 4 mm diameter and 18mm length within 2 cm proximal to carotid bulb.

We depend not only on the noninvasive color-flow echo Doppler scanning that is highly sensitive for neck fistulae but also M R A that confirm the diagnosis. This acquired fistula is similar to the Rod-shaped congenital type [6] but toursous and single branched with no other interconnected trunks.

There are no available definitive standard surgical or endovascular techniques to manage the carotid-jugular fistulas. Detachable balloons, stenting, coiling or operative ligations had been documented as treatment options [7]. Our patient preferred the surgical option, there by anatomic and pathophysiological aspects of the endovascular therapy as the use of plugs or coils and/or glue is to be avoided at the level of internal carotid artery because of the association with a high risk of pulmonary and cerebral embolization moreover, the use of carotid stent cannot ensure the effective closure of the communication. The ideal approach was to expose the fistula and double repair of both opening without any interruption of vascular continuity of both vessels. We did not dissect the CCA entirely and only IJV was controlled by elastic loop (Figure 2). So the unexpected complication did not occur neither immediately postoperatively nor on the first or the second day. Also, there were no evidence to explain the neurological deficits that happened on the third day in the light of normal laboratory investigation, CT brain and color coated duplex ultrasonography on the carotid system.

In general arteriovenous fistulae may impair the remote circulation; their effects at the microcirculatory level could not well understood yet [8]. Cerebral Hyperperfusion Syndrome (CHS) has been defined as a neurological deficit that occurs after cerebral revascularization after thromboembolism is ruled out as the underlying etiology [9]. This syndrome commonly presents with sudden onset focal neurological deficit, headache, seizure, and systemic hypertension after procedures that result in cerebral revascularization. The commonest imaging finding includes gyral thickening with obliteration of the sulcus spaces (commonly in the parieto-occipital lobes) and intracranial hemorrhage [10]. The first report on CHS after CAS was published by Schoser et al. [11] and reported by numerous publications ranges at 0.4% to 14% following carotid interventions (CEA, CAS and intracranial) and other various procedures such as vertebral and subclavian angioplasty with stenting, aorto-carotid bypass surgery, extracranial-intracranial and carotid-subclavian bypass surgeries, in nominate endarterectomy, dural arteriovenous fistula embolization, arteriovenous malformation resection, and clipping of giant ICA aneurysm. In this case CHS mechanism could be explained by increased flow through formerly increased flow through normal caliber arteries due to flow rerouting into cerebral parenchyma. CHS [12] had been classified CHS into three types based on their clinical and imaging features: Acute Focal Edema (AFE), Acute Intracranial Hemorrhage (AIH) and Vasogenic Edema (VE) in the affected cerebral territory with no change on diffusion-weighted images. Vasogenic Edema that is transient and reversible; could be pathology in our case due to normal imaging in addition to

that use of pharmacological agents and lack of periprocedural blood pressure control may simulate the clinical and imaging features of CHS.

CHS might develop at any time from immediately after the procedure to up to a month later, but most patients develop symptoms within the first few days. Ogasawa et al. [13] reported occurrence of CHS peaking on 6th post op day after CEA and 12 h after CAS. In our case the neurological deficits developed at the third day.

There is no randomized control trial document the optimized management protocol for CHS patients. However, blood pressure control with target blood pressure within 20% to 30% under baseline and cerebral anti-edematous measures as hypertonic saline, hyperventilation and steroids have been shown to be effective, anticonvulsants to treat seizures. These measures had been addressed to our case [14].

Despite the short duration of presence of the CJF in the present case, the patient, it had a high-flow arteriovenous fistula that had resulted in a state of a hypoperfusion in the ipsilateral cerebral hemisphere. By the surgical approaches, a sudden restoration of blood flow to a hypoperfused cerebral hemisphere with a disturbed auto regulation.

At the end we recommend that all carotid jugular fistula should be treated either surgically or endovascular therapy avoiding further complications such as infection, thrombosis, and arterial embolization. Ultimately, instruct patients accordingly at the time of discharge should to be aware about possibility of a delayed CHS. We should follow the following in standard IJV catheterization technique; limiting head rotation to avoid anatomic overlapping between the vessels, use of ultrasound-guided cannulation and a dilator should not be advanced over a kinked guidewire since this might change the direction of the dilator.

Conclusion

Carotid jugular fistula is a rare complication of IJV catheterization. Cerebral hyperperfusion syndrome could happen after CJF elimination whatever its duration that can be reversible with early recognition and proper management.

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